that the whole sac was any smaller. The development of what does appear to be a fine layer of calcium in the sac of the first case three years after treatment began is of interest from several points of view. In the first place calcification and syphilitic lesions of the aorta do not, as a rule, go together. One sees very few aneurysms at autopsy showing any evidence of calcification in their walls. It would be very interesting to know whether the so-called spontaneously healed aneurysm, which we know occurs, has calcium deposits in its wall, and also whether a positive history of syphilis is present. One is tempted to ask if in our case this deposit bears any relation to or is an index of healing. At the present time, however, we are able to do little more than note its occurrence.

We must admit that we are favorably impressed with the results so far observed, and therefore feel justified in recommending more active antisyphilitic treatment for aortic aneurysm.

BIBLIOGRAPHY.

1. Arnold: Am. Jour. Med. Sc., 1902, cxxii, 72.
2. Guthrie: Jour. Am. Med. Assn., 1919, lxxiii, 186.
3. Klotz: Jour. Path. and Bacteriol., 1913, xviii, 259.
4. Longcope: Arch. Int. Med., 1913, xi, 14.
5. Osler: Allbutt's System of Medicine, vi, 620. Osler: Modern Medicine, iv, 499.
6. Taylor, A. E.: Jour. Exp. Med., 1898, iii, 371.
7. Winternitz: Johns Hopkins Hosp. Bull., 1913, xxiv, 212.

THE SIGNIFICANCE OF YELLOW SPINAL FLUID.

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The occurrence of a yellow spinal fluid is sufficiently rare as to excite interest and sufficiently common to merit a brief consideration of its significance. The true syndrome of Froin[1] consists in (1) a spinal fluid which coagulates at once or upon standing—the consistency of the clot varying, but often so tenacious as to permit one to invert the tube; (2) a yellow coloration or xanthochromia; (3) a marked increase in cells and albumin. Froin[2] and all others who have studied this condition are agreed that the coloration is due to the presence of blood pigments chiefly bilirubin and the increased coagulability to an increase in fibrin. We shall not attempt to review the causative factors, mechanical and clinical, that bring about this departure from the usual pathological findings in spinal fluids but proceed to a consideration of the conditions in which it is encountered.

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[1] Gaz. des hôp., Paris, 1903.

[2] Syndrome de Coagulation Massive, Gaz. des hôp., 1903, p. 1587.

In a description of 100 cases Sprunt and Walker[3] state that the majority of cases showing yellow fluid present the complete syndrome but in 96 cases we have only had it occur in six instances. It appears that the true syndrome of Froin represents the highest development of a process in the spinal fluid of which the yellow coloration without the other essentials represents the earliest stage. Our 6 cases that presented the complete syndrome were 2 meningeal hemorrhages, 2 cases of tuberculous meningitis, and 2 of poliomyelitis.

In 95 cases of cord tumors and other chronic surgical conditions of the spinal cord which Elsberg and Rochefort[4] studied at the Neurological Institute, New York City, they found yellow fluid in 14 cases and spontaneous coagulation in 7 cases—a rather high proportion which would lead me to believe that disease of this kind tends to produce the conditions necessary for the development of the complete syndrome of Froin as stated by Mestrezat[5] to wit: (1) the existence of a closed cavity shut off from the general circulation in which the elements derived from blood accumulate (by transudation or hemorrhage); (2) an alteration (infectious or toxic) of the vessels of the cavity bringing about a transudation or a number of minute hemorrhages. In Dr. Elsberg's series of cases in which yellow fluid occurred, there were ten extramedullary tumors, two gummas, one varicose vein of the cord, and one neuritis of the cauda equina.

Our series of cases, 96 in all, represent the number of instances in which these cases have occurred in nine years of work of the Meningitis Division of the Health Department. There were 5801 fluids so that the percentage of cases in which yellow fluid occurred was about 1.6 per cent. Of these 96 cases, 60 have been tabulated and accompany this paper in the form of a table which is appended. Thirty-six cases were omitted from tabulation because the diagnosis was never confirmed definitely or there was lack of sufficient data. The chief interest of our series of cases lies in the fact that we see almost exclusively acute or subacute types of disease—which probably accounts for the small number of cases in which the syndrome of Froin occurred in all its manifestatins. Our cases were as follows: tuberculous meningitis 40, poliomyelitis 13, cerebral hemorrhage 3, meningeal hemorrhage 1, cord tumor 1, pachymeningitis 1, cerebrospinal meningitis 1.

Yellow fluids occurred in many cases of recovering epidemic meningitis but were omitted from consideration in this paper

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[3] Significance of Xanthochromia of the Cerebrospinal Fluid, Bull. Johns Hopkins Hosp., 1907, xxviii, 80.

[4] Xanthocromia and Other Changes in the Cerebrospinal Fluid, Jour. Am. Med. Assn., vol. lxviii.

[5] Le Liquide Cephalorachidien, Paris, 1912.

TABLE OF YELLOW FLUIDS.

No.	Amount, c.c.	Coagulation.	Albumin.	Globulin.	Fehling's.	Cells, per cent.	Diagnosis.
1	60	0	++1 solid	++ solid	+1	+ 95 (polys.)	Tb. meningitis.
2	15	+++	+++++	+++++	+1	++95 (monos.)	Poliomyelitis.
3	15	0	++++	+++	+	+95 (monos.)	Poliomyelitis.
4	10	0	++++	+++	++	+95 (monos.)	Tb. meningitis.
5	5	0	+++	++++	0	+90 (monos.)	Tb. meningitis.
6	8	0	+++	++	N.d.	None (?)	Poliomyelitis.
7	3	0	++	+++	N.d.	+	Tb. meningitis.
8	20	0	+++	+++	N.d.	+90 (monos.)	Tb. meningitis.
9	40	0	+	+	N.d.	Red blood cells	Meningeal hemorr.
10	8	0	+++	+++	N.d.	+95 (monos.)	Tb. meningitis.
11	3	0	++++	++++	N.d.	+95 (monos.)	Tb. meningitis.
12	8	0	+++	++++	+	+60 (monos.)	Poliomyelitis.
13	40	0	+++	+++	0	+50 (monos.)	Tb. meningitis.
14	70	0	+++	++	N.d.	90 (monos.)	Tb. meningitis.
15	10	0	++	N.d.	N.d.	Few cells	Pachymeningitis.
16	5	0	++	+++	N.d.	++	Poliomyelitis.
17	5	0	++++	++++	+1	+90 (monos.)	Tb. meningitis.
18	10	+	++++	++++	+1	+98 (monos.)	Tb. meningitis.
19	40	0	+++1	++++	+	+95 (monos.)	Tb. meningitis.
20	30	0	++++	++++	+1	++90 (monos.)	Tb. meningitis.
21	20	0	++++	++++	+	+90 (monos.)	Tb. meningitis.
22	20	0	++++	++++	++1	+90 (monos.)	Tb. meningitis.
23	30	0	++++	++++	++1	+80 (monos.)	Tb. meningitis.
24	25	0	++++	++++	++1	+95 (monos.)	Poliomyelitis.
25	15	0	++++	++++	+1	+70 (polys.)	Tb. meningitis.
26	10	0	++++	++++	++	+90 (monos.)	Tb. meningitis.
27	25	0	+++++	+++++	+	+90 (monos.)	Tb. meningitis.
28	35	0	++++	++++	+1	+90 (monos.)	Tb. meningitis.
29	5	0	++++	++++	++1	+90 (monos.)	Tb. meningitis.
30	5	+	N.d.	++++	–	Few	Meningeal hemorr.
31	2	0	++++	++++	+1	+	Tb. meningitis.
32	5	0	++++	+++	+++	Slight + 90 (monos.)	Cerebral hemorrhage.
33	30	0	+	+	+++	++	E. C. S. W.
34	35	0	++++	++++	+1	+90 (monos.)	Tb. meningitis.
35	20	++	+++++	++++	++	+70 (monos.)	Meningeal hemor.
36	20	0	++++	++++	+	+mod. 90 (mon.)	Tb. meningitis.
37	20	0	++++	++++	+	+80 (monos.)	Tb. meningitis.
38	3	..	++++	++++	N.d.	N.d.	Poliomyelitis.
39	20	0	+++	+++1	+++	+60 (monos.)	Poliomyelitis.
40	10	0	++++	++++	+1	+95 (monos.)	Tb. meningitis.
41	20	0	++++	++++	0	+98 (monos.)	Tb. meningitis.
42	5	0	+++++	+++++	+	+89 (monos.)	Tb. meningitis.
43[6]	30	0	++++	++++	+++	Moderate +	Poliomyelitis.
44[7]	25	0	++++	++++	+++	+ monos.	Poliomyelitis.
45[8]	40	0	++++	++++	+	+90 (monos.)	Tb. meningitis.
46	30	0	++++	++++	+	+90 (monos.)	Tb. meningitis.
47	2	0	++++	N.d.	N.d.	+	Tb. meningitis.
48	3	0	++++	+++	+1	+90 (monos.)	Tb. meningitis.
49	10	0	+++	+++	+	+99 (monos.)	Tb. meningitis.
50	20	0	+++	+++1	+	+95 (monos.)	Tb. meningitis.
51	40	0	+++	+++	+++	+90 (monos.)	Poliomyelitis.
52	20	0	+++	++1	+	+mod. 95	Tb. meningitis.
53	15	0	+++	+++	+++	N.d.	Cerebral hemorrhage.
54	10	0	++++	+++	+++	+90 (monos.)	Tb. meningitis.
55	30	0	++1	++1	+1	+50 (monos.)	Poliomyelitis.
56	60	0	+++	+++	+1	+99 (monos.)	Tb. meningitis.
57	15	0	++++	++++	+	+95 (monos.)	Tb. meningitis.
58	15	0	++++	++++	++++	+75 (monos.)	Poliomyelitis.
59	30	0	+++	++	++	+90 (monos.)	Tb. meningitis.
60	30	0	++	+++	+	+99 (monos.)	Tb. meningitis.

[6] Fluid No. 43, gold chloride curve, 1111, 232100.
[7] Fluid No. 44, gold chloride curve, 111, 22332210.
[8] Fluid No. 45, gold chloride curve, 111, 2322100.

N.d., not done.

Summary:

Tuberculous meningitis	40
Poliomyelitis	13
Cerebral hemorrhage	3
Epidemic meningitis	1
Pachymeningitis	1
Syringomyelia	1
Meningeal hemorrhage	1
Total	60

because the condition did not occur spontaneously but after the administration of serum, which, however, we do not consider the cause of the xanthochromia.

Mestrezat[9] and many of the French writers have pointed out that the occurrence of a yellow fluid is not uncommon in tuberculous meningitis, yet it forms a very striking fact that it occurs in over 66⅔ per cent. of our cases. Neal and DuBois[10] have already pointed out that yellow fluids occur too in poliomyelitis.

It had long been my clinical observation which is confirmed by these figures that given a patient with meningeal symptoms who on lumbar puncture shows yellow spinal fluid one should be inclined to a diagnosis of tuberculous meningitis or poliomyelitis—this evidence, of course, being by no means conclusive but merely a factor to be considered in the other points of diagnosis which further study of the spinal fluid usually decides.

The spinal fluid withdrawn in these cases has varied in amount from 3 c.c. to 50 c.c. The color has ranged from the faintest of yellow tints to the deepest canary. The albumin and globulin have been markedly increased in the majority of the cases. The sugar reactions as shown by the reduction of Fehling's solution has varied with the underlying etiological infection. In three instances colloidal gold curves were performed and these are included in the table.

RÉSUMÉ. (1) Yellow spinal fluid occurs in a wide range of diseases of the spinal cord and meninges; (2) the complete syndrome of Froin is comparatively rare in its occurrence; (3) in acute or subacute conditions the presence of yellow fluid strongly suggests the probable diagnosis of tuberculous meningitis or poliomyelitis.

THE OCCURRENCE OF GLYCOSURIA IN MUSHROOM-POISONING, WITH THE REPORT OF FIVE CASES IN WHICH A MILD NEPHRITIS AND A RENAL GLYCOSURIA WERE THE PERSISTENT AND PREDOMINATING FEATURES.

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ALTHOUGH the history of mushroom-poisoning reaches back to the times of Babylonia and ancient Rome, most of the reports, even the more recent, give but little clinical details and no accurate account of postmortem findings. In reviewing the literature one is also impressed by the few reports of cases that have ended in recovery. Although recent investigators, as Clark, Marshal and

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[9] Same.

[10] The Diagnosis of Poliomyelitis, AM. JOUR. MED. SC., September, 1916.